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ORIGINAL ARTICLES

HEART BLOCK*

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The mechanisms associated with the heart beat have for years been sources of never ending interest to the Physiologist. The causes of interference with these mechanisms have formed fascinating subjects for many Pathologists. The inter-relationship between the two is the field of the Laboratory Cardiologist, while the unfortunate humans exhibiting abnormal cardiac mechanisms turn for assistance to the clinical Cardiologist and even more frequently to the harried general practitioner. It is well therefore that this man of universal Medicine should occasionally pause to review his knowledge, and perhaps add to his knowledge of the abnormal cardiac behavior which he meets so constantly. Some of these abnormal behaviors grouped under the heading of Heart Block will be briefly scanned this evening.

Perhaps for the sake of clearness, some basic facts had better be recalled. The human heart is evolved from an embryonic organ containing a sinus venosus. This is later submerged in the musculature forming the posterior part of the right auricle. Here, in the *sulcus terminalis* is a club-shaped mass surrounded by a coronary vascular circle. This mass, the Sino-Auricular Node sends a few strands up the superior vena cava, its lower end terminating half way between this vessel and the Coronary Sinus. The remainder of the original sinus tissue is condensed into the auricular part of the Auriculo-Ventricular Node which lies in the region of the Coronary Sinus. The ventricular portion of this node is derived from the tissue of the auricular canal.

From the Auriculo-Ventricular Node a round bundle passes across the Auriculo-Ventricular Septum and courses downwards and forwards until reaching the interventricular septum it in turn divides into right and left branches. These branches in turn spread an extensive aborization throughout their respective ventricles on the inner surface of the ventricular walls.

The Sino-Auricular Node becomes active or electrically negative before any other region, under normal conditions. For this reason it is called the Pacemaker. If it be depressed or destroyed, the irritability of the Auriculo-Ventricular Node supervenes and it in turn becomes pacemaker, generally at the Coronary portion. If this in turn becomes interfered with the progressive Pacemakers thereafter are, in order of precedence, the ventricular portion the His bundle, or the Branches.

It is doubtful if any center below the Branches can effectually dominate the rhythm of the heart. Their importance lies in that they serve to disturb some dominating rhythm as in the production of extrasystoles.

No differentiated conduction paths have been shown to exist in the auricle. Impulses from the Sino-Auricular Node spread radially over ordinary auricular tissue at a speed rate of 600 to 1200 mm. per second according to the determinations of Lewis Meakins and White. Bachmann has shown that impulses probably pass to the left auricle by the inter-auricular band. Impulses reach the Auriculo-Ventricular Node via a simple auricular tissue and of course the bundle and its branches continue the conduction to the Ventricles.

Wiggers found the transmission rate to be 200 mm. per second in the Auriculo-Ventricular Node and 3000 to 5000 mm. per second in the bundle and its branches. He states that ventricular muscle conduction is at a rate of 400 mm. per second. The evident delay at the Auriculo-Ventricular Node insures stimulation in the furthest reaches of the Auricle, the Auricular Appendices, before the ventricles become excited. The rate of transmission through the bundle, insures the arrival of the impulses at the terminal aborizations at approximately the same time.

Practically this means that the stimulation from the Sino-Auricular Node to the Auriculo-Ventricular Node occupies about 0.10 second, that there is

*—Delivered before the Providence Medical Association, April 1, 1929.

a delay of 0.05 second in the Auriculo-Ventricular Node and that the time taken from that point to the terminal aborization is 0.01 second.

Cullis and Dixon applied Cocaine to the conducting system of dogs. The nerve fibers were paralyzed but conduction was unhindered. We may therefore deduce from this evidence that transmission is myogenic rather than neurogenic in nature.

Electrocardiographically transmission from the Sino-Auricular Node to the end of the Bundle ramifications is measured by the P-R interval. The P wave represents spread of negativity in the auricles, the beginning of the R stroke indicates the beginning of ventricular activity. The gap between the P wave and the R represents a delay of transmission through the Auriculo-Ventricular Node, the bundle and its branches.

The nerve supply of the heart is derived from the medulla through the vagus trunks and from the upper segments of the thoracic cord through the sympathetic chain. The supply is essentially homolateral though there is some overlapping of the right fibers on the anterior aspect of the ventricle. When outside of the Auriculo-Ventricular Node and Bundle it is impossible to decide whether the fibers belong to the Vagus or Sympathetic Systems.

Stimulation of the right vagus nerve slows the heart without any alteration in conduction time. Stimulation of the left vagus causes a marked ventricular slowing without altering the auricular rate. This means that rhythm production in the Sino-Auricular Node is under right vagus control whereas the left vagus is more concerned with increasing auriculo-ventricular conduction time.

Strong stimulation of the vagus, principally the left, but at times also the right, and certainly in the event of simultaneous stimulation of both vagi, may cause the seat of impulse formation to move progressively to regions of lower automaticity. A complete block of impulses across the Auriculo-Ventricular tissue may occur. When the ventricle escapes from such inhibition and establishes a rhythm of its own, the impulses may originate in the main bundle or one of its branches, in which case one ventricular beat precedes the other.

The right accelerator nerve chiefly affects the S-A Node while the left chiefly affects the A-V Node and therefore stimulation of the right stellate ganglion increases auricular and ventricular rate without altering conduction time whereas stimulation of the left markedly shortens the P-R interval as well as increases the rate.

Nature of Heart Block

This is two fold, firstly an Anatomical Disconuity as when the tract of conducting tissue is cut across by disease or experimental incision. Secondly, Physiologically as when through disease processes of Chemical poisons toxic absorption results in depressed excitability of the conducting tissue.

Clinically block can occur at any level and expresses itself in all grades from Sino-Auricular Standstill to Arborization Block.

If we agree with Englemann that conductivity, excitability and contractility are three separate and distinct functions, then we can explain a missed response of the ventricle in one of three ways. However the separateness of these functions has not received any general assent. Lewis finds that evidence accumulates steadily against this theory.

Latency is the hesitancy of muscle to respond to stimulation over a measurable interval. Erlanger, Straub and Mobitz consider latency as an important part of Heart Block. Lewis however seems convinced that there are as yet entirely insufficient data from which to argue either one way or the other. He considers the phenomena involved thus far too complex to be understood properly.

Altered Transmission Rate Between Points is another explanation of increasing P-R intervals. Transmission through auricular muscle widens—

1. When the heart rate is sufficiently raised.
2. When it is under the influence of Strophanthin or Quinidine.
3. Experimentally when the heart is perfused with a nutrient fluid, more acid than normal, or when the junctional tissues are cooled.

This of course inevitably brings into discussion what is known as The Partial Refractory Period. When the mammalian auricle is driven at excessive speed, the ventricle fails to follow suit and breaks eventually into an unstable 2:1 response. The ventricle is thus guarded from excessive impulses by tissue having a relatively long refractory period. However before the ventricle fails fully to respond there is a stage of progressive widening

of transmission intervals due to the development of a state of partial refractoriness. This means that the response to stimulation is not simultaneous and unanimous in the neighboring fibers and the wave wandering through sinuous channels is late in reaching its destination. When the rate of response has been increased beyond a certain point the transmission intervals, previously of unchanged length, begin to widen out progressively. This theory will account for—

1. The phenomena of widening intervals.
2. The curious arrangment of intervals around a dropped beat.
3. The actual dropped beat itself and the close association between Block and Alteration.
4. Auricular Flutter and its mechanism.

Partial Refractoriness is either partly or wholly responsible in the block produced by toxins directly on the muscle such as the Digitalis bodies, Quinidine, Veratrin, etc.

Decrement is the continuous decline in strength of an impulse as it travels through defective tissue. Drury and Andrus have offered some preliminary evidence that this phenomenon is one means of explaining certain features of block.

Carter and Dieuaide have suggested that in the block of Hay's type in which ventricular beats are dropped without preliminary lengthening of the P-R intervals, the defect is in the bundle rather than in the A-V Node.

It is well also to recall here that when block is already present, Vagus action increases it.

Etiology

The causes may be grouped under the headings Therapeutic, Toxic, Mechanical.

Therapeutic

Commonly the Digitalis bodies, Quinidine, Aconite, and in susceptible persons Morphine and the Salicylates. Perhaps Aspirin more frequently than others.

Toxic and Mechanical

Diphtheria produces an especially acute and fulminating type of block. It may affect the vagus centrally or peripherally in the S-A or A-V nodes. By its well known degenerative myocardial action it may also exert an effect whose nature is mechanical as well as toxic.

Rheumatic Fever even in mild cases may exert a toxic effect on nodal tissue and a mechanical effect through its inflammatory reactions—edema, cellular infiltrations, Aschoff bodies, etc.

Syphilis in the early stages by toxic action may produce toxic block similar to Diphtheria and Rheumatic Fever. Later by gunmata and fibrous tissue proliferation mechanical block frequently occurs.

The Bacteriemias and Septicemias, including Staphylococcal, Streptococcal, Gonococcal, Pneumococcal, Influenzal and Typhoid have also shown varying grades of block toxic and mechanical.

The exanthemata are at times offenders.

Anterior Poliomyelitis and Anaphylactic Shock both show the progressive type of Block known as Asphyxial block, so called from its identity with Lewis' Cat Asphyxiation experiments.

Mechanical

Occlusion of the Arterial supply to the Bundle and its Branches Aneurysmal dilatations of the Coronary vessels pressing on the conduction system, Tumors, any fibrosing, calcifying, sclerosing, infiltrating or inflammatory reaction directly interfering with the transmission of stimuli across the normal pathways of conduction.

A possible Classification of the Grades and Types of Heart Block might be as follows:

1. Sino-Auricular Standstill. 2. Auriculo-Ventricular Block. *Partial*—1st Degree, involving only lengthening of the P-R interval; 2nd Degree, involving progressive widening of the P-R interval with dropped beats. This occurs mostly in cycles. 3rd Degree—Simple ratios, 2:1, 3:1, 4:1, and even greater. *Complete.* Showing auricles and ventricles beating on their own rhythms independently of one another. Complete Block includes *Congenital Block.* A-V Block partial is often Digitalis effect. 3. Bundle Branch Block. 4. Arborization Block. 5. Auricular Flutter.

Sino-Auricular Standstill

Resnik in 1923 contended that Sino-Auricular Block was a misnomer. His electrocardiograph records show complete absence of demonstrable Sinus activity in the long pauses. He concluded that it was merely one of the forms of Sinus Arrythmia.

Sino-Auricular Standstill shows itself as an occasional dropped beat, or at times a group of dropped beats with auricular as well as ventricular silence. These droppings may regularly recur. Again it may show itself as a sudden precise halving of the normal rate, in which case exercise, excitement or emotion may restore suddenly the original rate and thereafter with continuation of the exercise, etc., will steadily increase it. The stimulus removed, the rate gradually slows to 70 or 60 and then precisely halves again. There may be profound and continued slow action of the entire heart—and finally there may be a complete Sino-Auricular Standstill with onset of Nodal rhythm.

Without doubt Sino-Auricular Standstill is a vagus effect as it is always abolishable with Atropine.

The toxins of Rheumatic Fever, Diphtheria, the Septicemias and Bacteriemias have produced it rather strikingly.

Cockayne and Hamburger have reported cases of Sino-Auricular standstill apparently produced by the toxin of Influenza. Laslett reported another striking case in which the heart went into total Standstill for from 4 to 8 seconds.

Neuberger reported a case of standstill occurring repeatedly in a man during the act of defecation. Autopsy showed an Aneurysm upon the basilar artery producing pressure upon the medullary centers during sudden rises in blood pressure from any cause.

Wiggers thinks the condition is not nearly so rare as has been reported and he questions its pathological significance pointing to the large number of undoubtedly healthy persons who have it.

One is inclined to look upon Sino-Auricular Standstill as an interesting fairly frequent but clinically rather unimportant affair, having no essential bearing upon an individual's future, being significant of vagotonia and therapeutically very much of a nonentity.

Auriculo-Ventricular Block

In contrast to Sino-Auricular Standstill Auriculo-Ventricular Block is of decided interest to everybody concerned and of course has a most important reference to the future of its victim.

The incidence of first degree block is common, perhaps more as a Digitalis effect than as a toxic effect. This may synchronize with the subjective symptoms of nausea and vomiting. *Per se* first degree block has no subjective symptoms and beyond the slowing of the pulse and a reduplication of the first heart sound there are usually no signs. It is well however to recall that in Mitral Stenosis cases which have usually shown the characteristic presystolic rumble and thrill there is a change in the presence of first degree block to a rumble and thrill of mid-diastolic or early diastolic timing.

Of the Infectious diseases Diphtheria and Rheumatic Fever are probably the most frequent offenders but other infectious diseases offend often enough to be noticed—and I must mention here one interesting case I saw in the wards of the Johns Hopkins Hospital. He was a white man, seriously ill with Typhoid whose P-R interval steadily climbed to 0.36. He did not increase his block further and after many weeks he eventually recovered a normal conduction time.

Myocardial Degenerations have their share in the production of first degree block as for example the case of an elderly negro attending the J. H. H. Dispensary. This man has shown a P-R interval varying for over a year between 0.22 and 0.26 without advance or retrogression in degree of block. Cary Coombs, in his Text Book "Rheumatic Heart Disease" refers to a man with Chronic Cardiac Rheumatism who had a partial block for years without changing in character or degree. In such cases he suggests that there is probably a permanent lesion involving the Artery to the Bundle. He states that Block on a Rheumatic basis while frequent enough is seldom more than trivial in degree.

Second Degree Block is merely a stage in advance of first degree block in that the P-R intervals progressively increase until one auricular contraction fails to find a ventricular response. The P-R interval immediately thereafter shortens and then there is a progressive widening until the dropped beat again occurs.

Etiologically identical with first degree block it differs objectively in that there are periods of occasionally recurring ventricular silences, the pause at times being exactly equal to two rhythmic beats but generally falling somewhat short of this. The radial pulse before the dropped beat as well as after it shows a tendency to lengthening intervals.

Lewis found that with exercise and the inhalation of amyl nitrite the pulse quickens and be-

comes regular. With rest the irregularity re-appears and the first sign of its return is the occurrence of an unusually long ventricular pause.

As the number of the dropped beats increases in frequency there is a tendency to regularity in the grouping of beats and intermissions. Even at this stage, exercise and Amyl Nitrite will abolish the block, but of course only temporarily.

Second degree block has another feature, called at times the Block of Hay's type in which there is a sudden dropping of ventricular responses without the feature of increasingly progressing P-R intervals.

Third Degree Block immediately follows on late Second Degree Block. It is characterized objectively by ratios such as 2:1, 3:1, 4:1 and so on. It is to be suspected in any patient in whom the heart beats regularly, and in whom the rate lies between 40 and 50 per minute.

Lewis again discussing Mitral Stenosis points to the two presystolic thrills and rumbles that accompany one ventricular beat in the 2:1 ratio and of course the higher the ratio of block the greater number of thrills and rumbles to the ventricular beat.

In block as high as 2:1 exercise and Amyl Nitrite may serve temporarily to remove the block. The former while doubtless experimentally possible is surely not to be tried indiscriminately. There is of course no therapeutic gain to be achieved from it to say the least.

THE ZONE OF DANGER. THE ZONE OF VENTRICULAR STANDSTILL LIES IN THIS NEIGHBORHOOD. Third degree Block is notoriously unstable and advances readily to the next order of procedure which is complete block with Ventricular Standstill. The Bundle may then take on a Pacemaker, and if it does within reasonable time, well and good. The obvious danger that standstill may remain standstill is the matter of gravest concern both to physician and patient. Even when the bundle does develop a Pacemaker there is generally a good deal of struggle for control at least temporarily between the new center and the centers of higher rhythmicity, involving of course many attacks of standstill due to the refractory A-V Node.

The "Zone of Danger" has the Stokes Adams Syndrome as its characteristic manifestation and this well known syndrome varies in intensity of action directly in proportion to the extent and duration of Cerebral Anemia. I would like here to describe some of my own cases illustrating this in several ways.

Case 1: Man in early fifties, laborer, arterio-sclerotic seen in No. 5 Dispensary J. H. H. Complaints: Attacks vertigo, dimness of vision, nausea. During examination pulse dropped sharply from 66 to 33 for perhaps as long as 20 seconds and then resumed its usual rate.

During the attack he complained of nausea, and dizziness and he firmly grasped the chair on which he was sitting saying he could hardly see and feared he might fall. His pupils were measurably dilated at this time. His ordinary pallor became intensified without a trace of cyanosis, his breathing increased in rate and depth.

Objectively he showed in between attacks the ordinary signs of myocardial enlargement with some edema in the extremities, and some dyspnoea on ordinary exertion. His blood pressure in both phases was within normal limits.

He remained a while in Hospital, reverted to normal conduction time and went back to his home outside the State. Nothing has recently been heard of him.

Case 2: New England housekeeper, 76 years, a diabetic of some years standing and an advanced Arterio-sclerotic.

Complaint: Several abrupt and heavy falls with complete loss of consciousness. Attacks coming on with devastating suddenness.

When I saw her she was lying in bed. She extended to greet me a trembling hand. Her grip in both hands, usually firm, was feeble. Her speech usually crisp and decisive was halting. She said she was too weak to move about in bed without assistance and found it most difficult to move either arms or legs.

When I started to examine her, her pulse rate was 80. It suddenly dropped to 20. At the time she was in the midst of a sentence. She stopped the sentence suddenly. Her face became markedly blanched, her pupils widely dilated and she started a stertorous respiration. There was some slight twitching of the facial muscles. I should judge that this lasted perhaps twenty seconds when the pulse suddenly reverted to 80. Consciousness immediately returned and she finished her sentence. She seemed entirely unaware of her attack of

Stokes-Adams. I remained in the house for an hour thereafter during which time she had five such attacks.

She is now up and about with a pulse rate of 40. She has had no attacks for nine months, but for six weeks she went through many Stokes-Adams fits and finally developed a complete A-V dissociation. She is attending to very much restricted duties. She becomes dyspnoeic and has some precordial pain and edema if she overdoes. Living quietly she has no subjective symptoms.

Case 3: Elderly Sea Captain, advanced arteriosclerotic with myocardial degeneration, dyspnoea, and tachycardia on exertion, some pitting oedema in the extremities, heart quite enlarged, occasional gallop rhythm, B. P. within normal limits in both phases. One day without warning he fell heavily to the floor in a Stokes-Adams fit. By the time I reached him 15 minutes had elapsed. I found him with a feeble alternating pulse about 90. I was in the act of preparing a hypo of Adrenalin for him when he announced that he was dying. His heart showed complete ventricular standstill for about 15 to 20 seconds. He slumped back in his easy chair, his pallor was marked, his pupils dilated, his breath was stertorous, and twitchings were beginning in his facial muscles when the ventricle started up again at its usual rate.

Congenital Block is really part of Auriculo-Ventricular Block. It is usually associated with defective development of the Auriculo-Ventricular Bundle. As a result its victims subsist on an idioventricular rhythm.

Carter and Howland reported 12 such cases. Some of them were recognized as early as the twelfth day while others remained undetected till the twentieth year.

Aylward in the British Medical Journal Vol. 1., 1928, reports two cases of congenital block in one family. Both children show no other defects and their family history is excellent.

All these youngsters were apparently unhampered by this damage, running around actively with pulse rates in the neighborhood of forty.

This of course goes to show that the thing of the greatest importance is the health of the myocardium and only to a lesser extent the integrity of the conduction pathways.

Therapeutic Block should also be considered under the heading of A-V Block. Though, as previously mentioned, therapeutic block has occurred during the administration of Aconite, Veratrin, the Salicylates, etc., it is an accidental and often alarming affair. On the other hand therapeutic block of the A-V node is as all of you know, the most useful and valuable method of treatment when a fibrillating auricle is showering stimuli in excess upon the A-V Node. This of course is an affair of Digitalis and its family. When carried beyond therapeutic necessities it progresses beyond partial block into even greater grades. There is however so much involved in this subject that it is of course out of place in a paper such as this. Those interested can do no better than read the masterly work of Canby Robinson on Digitalis.

Last June in the American Heart Journal John T. King published a paper on the clinical diagnosis of Bundle Branch Block. His standards for its recognition were:

1. Visible Bifid Thrust.
2. Palpable Bifid Thrust.
3. Feeble heart sounds with a sound and an asynchronous murmur accompanying the two elements of the systolic thrust.

It was my good fortune to see two of Dr. King's nine cases, one in the ward and one in the Dispensary. Since the publication of his paper I have been able to recognize by these standards two other cases in our Dispensary. I have also reviewed and similarly recognized by these signs an old private patient whose diagnosis previously rested on EKG findings solely.

Although the bifid thrust presents little difficulty in recognition the laying across the chest of a piece of paper or a cloth materially facilitates this recognition.

In one case I was able to recognize Waldorp's sign — a reduplication of both first and second sounds.

All the cases I saw have been on a basis of Senile Myocardial degeneration as were eight out of nine in King's series. One other showed a history of Rheumatic Fever and one gave a history of Tonsillitis but the bearing of these diseases on the B. B. B. was most doubtful.

Rohmer in a series of studies of Diphtheria hearts ante mortem reported variations in the ventricular complexes suggesting conduction disturbances in the bundle branches.

Electrocardiographically the standards are:

1. Widening of the QRS complex beyond 0.10 seconds.

2. Notching of the QRS complex.

3. T wave in a direction opposite the main ventricular deflexion.

4. Dextrogram or Levogram predominant.

Carter's 20 cases showed 19 right bundle blocks, and one left.

King's 9 cases were all right blocks.

The 5 cases I saw were all right blocks.

It is assumed that the proportion is somewhere about ten right blocks to one left.

Most cases of B. B. B. are associated with a blood pressure either within normal limits or somewhat below. Certainly the ventricle cannot have as great a power while one is lagging as would be the case with both working synchronously. However some cases with hypertension have been noticed and the difficulty arises in distinguishing the sounds of B. B. B. from presystolic gallop rhythm. The bifid visible and palpable thrust will usually be sufficiently distinct.

I have incidentally recently seen a record where B. B. B. occurred paroxysmally.

Auricular Flutter

In this condition we find one continuous wave circulating around the openings of the superior and inferior vena cava. This differs from the ordinary contraction wave in that it does not have any radiation. It reminds one of an animal chasing its tail. It passes back over the same pathway again and again. It produces a very fast and feeble auricular beat varying from 150 to 360 per minute. The time elapsed in the completion of one circuit varies with its length and also with the rate of conduction. There is never any real diastolic auricular pause and thus the auricle gets no rest. There is of course at each circuit an impulse sent to the A-V node but owing to the phenomenon of Partial Refractoriness which we previously considered many impulses do not get through. Ordinarily Auricular Flutter is a 2:1 block though other ratios are seen. Regularity or irregularity in ventricular response depends upon the sensitivity of the A-V node.

Although flutter may occur in fleeting attacks it is more customarily a matter of months or years. Rheumatic Fever, Syphilis, and Influenza have been known to be etiological factors but in the vast majority of cases it may be attributed to Myocardial Degeneration and it is seen usually in persons of middle or advanced years.

There does not seem to be any characteristic morbid anatomy.

Flutter patients subjectively describe the onset of this rhythm as accompanied by palpitation and perhaps even by fainting. They may speak of several attacks antedating the final one of which they cannot rid themselves. During periods of excitement or emotion the ventricle may respond to every auricular impulse. The patient is then temporarily conscious of the frequency of the beat. With such speedy ventricular action however consciousness is rarely retained. In this eventuality vagus pressure is often of material assistance in slowing things up.

Confusion between Paroxysmal Tachycardia and Flutter may arise but if we remember that commonly Paroxysmal Tachycardias show ventricular rates of 180 to 220 per minute and that auricular rates surpassing 360 are unknown the matter is not so difficult and it is at all events of rather slight practical importance. In the matter of duration Paroxysmal Tachycardias do not last beyond 14 days while flutters last for the most part over months and years.

Flutter at times shows a feature characterized by irregular responses of the Ventricle to Auricular Stimulation. At this time confusion with Auricular Fibrillation is commonly made. During such times, however, exercise of even slight grade will accentuate ventricular action and induce perfect regularity of the pulse in a 2:1 Block.

Treatment

Sino-Auricular Standstill

This is seldom regarded as a therapeutic affair of much interest but should the symptoms arising from it be troublesome it can be easily controlled by Atropine in average dosage.

Auriculo-Ventricular Block

Treatment for this condition is divided by Hirschfelder into three phases:

1. To bring about retrogression of the lesion in the bundle.

2. To remove as many factors as possible which tend to increase the block.

3. To increase the irritability and rhythmicity of Ventricular muscle so as to shorten the periods of stoppage and to increase the rate of the ventricles.

N. B. I think he might have added a fourth phase: To increase the likelihood of the rapid development of new areas of sensitivity or negativity for the purpose of Pacemaking in emergencies.

The first phase of the Hirschfelder system is directed towards luetic lesions in the bundle. In many instances, particularly in early lues this results favorably. In later lues increase in block has at times followed mixed anti-luetic treatment, perhaps due to the depressing effect of K salts on the A-V node. This however should not cause anyone to hesitate about the application of anti-luetic treatment in all cases where it is indicated.

Where definite sclerosis has occurred little can be accomplished in a therapeutic way.

The remaining phases of the Hirschfelder system are directed to increasing the irritability of the A-V node and the potentially pacemaking tissues. Simple rest is at times surprisingly effective but generally more active. Treatment is called for and this is generally of a drug nature. Among the useful drugs having a bearing on A-V Block are: Atropine, Adrenalin, Nitroglycerin and the Nitrites, Thyroid Extract, Barium Chloride.

Atropine

Where block is "Vagus Effect" it is the drug par excellence. Even where the block is not vagal it may be possible by Atropine to prevent any subsequent vagus action which can always enhance a block regardless of its nature.

Adrenalin

This is the drug of emergency. Injected directly into the Myocardium it frequently abolishes ventricular standstill. Partial block may be likewise abolished by subcutaneous injections of Adrenalin. The effect is generally good for a few hours. There is no evidence pointing to any permanently beneficial effect either in the use of Adrenalin or its relative Ephedrin.

A subcutaneous injection of Adrenalin during complete standstill is of course unlikely to accomplish anything because ventricular standstill is synonymous with circulatory standstill.

Nitroglycerin and the Nitrites

These drugs, by producing vascular dilatation may stimulate an automatic increase in pulse rate to maintain the circulation. Amyl Nitrite inhaled works rather rapidly, but Nitroglycerin takes from 30 minutes to 3 hours to get in its action and it seems unable to influence the recurrence of attacks.

Thyroid Extract

E. H. Drake of Portland, Maine, after using 480 mgms. Barium Chloride without effect in Stokes-Adams attacks supplemented his treatment with Thyroid Extract beginning in doses of 3 grains daily. After 2 weeks he stepped it up to 10 grains daily for 5 days until nausea, vomiting and hot flashes began. He then stopped treatments. His patient has had no attacks for well over a year.

Barium Chloride

Rothberger and Winterberg showed that Barium and also Calcium increase ventricular irritability.

Von Egmond showed the same effect after traumatic experimental block with complete dissociation.

Cohn and Levine first used it for recurring attacks of ventricular standstill.

Herman and Ashman report favorable results in both transient complete block and also in block of any type showing syncopal attacks. They are convinced that Barium Chloride should be administered constantly in doses as high as 50 mgms. every four hours. They impress patients with the idea that life depends upon the presence of a constant minimum concentration of the drug in the heart muscle in order to keep irritable the ventricular pacemaker, in the case of total dissociation and the A-V node where the block is partial and the subject to recurrences of A-V nodal refractoriness, with ventricular standstill.

Strauss and Meyer concur entirely with this.

It must not be forgotten however that treatment in A-V block must be directed towards the damaged myocardium which frequently exhibits signs of greater or lesser failure. Rest and Digitalization are just as important here as elsewhere despite the trepidation of many clinicians. The words of Lewis here are very much to the point: "The increase of block should not deter Digitalis administration for it is not in itself detrimental

and where indicated should be given without restraint."

In Arborization Block as previously mentioned treatment is directed solely to the damaged myocardium.

Many clinicians have at times been impressed with the favorable action of Quinidine in average dosage on Bundle Branch Block.

The treatment of Auricular Flutter is preferably Digitalization and Digitalis is given until there is a definitely slowing action in the ventricles. It should thereafter be still further pushed until the Flutter is converted into Fibrillation. The drug is then withdrawn, whereupon normal sinus rhythm gradually supervenes. It is possible for flutter to return thereafter but when it does return there is usually a lapse of a rather long time.

Prognosis

Every case of heart block demands close observation over considerable time. During a period when fits are noted, if the feebleness of the patient and the frequency of the fits do not in themselves enforce rest in bed or at least confinement to the home it is important to remember with what catastrophic suddenness fits come on. Three seconds of standstill are sufficient for unconsciousness. It takes considerably more than three seconds to cross a street and with motor traffic at its present day speed the fate of a pedestrian in a Stokes-Adams attack may easily be one of violent demise.

It follows that if such a patient cannot be induced to stay at home in between attacks he should at least always be accompanied.

Many such persons are found dead in their beds, or elsewhere.

Most persons die within three years of their first Stokes-Adams attack.

In a few cases the first attack is the last.

Frequent recurrence of these attacks signifies gross myocardial damage in the majority of cases.

Heart block mild or severe is usually an evidence of myocardial damage generally well diffused throughout the heart.

In the cases where there is an occlusion of the artery to the bundle which is an end artery it is possible that this accident may occur unassociated with any generalized myocardial involvement and it is probable that the large number of those who carry complete dissociation for decades of years and yet seem to thrive have had a simple arterial occlusion as an etiological factor.

Where heart block is definitely on a luetic basis the prognosis is materially brighter than in those whose basis is degeneration or sclerosis. Specific treatment often permanently restores normal conduction.

A very large number of hearts which show delayed conduction on a basis of Influenzal, Pneumoccal or Typhoid toxemia entirely recover.

When defective conduction is on a Diphtheritic or Rheumatic basis there is a greater likelihood of permanence.

Block on any toxemic basis whatever is always to be regarded as an adverse element in the prognosis, when all elements are being summed up. It may be definitely regarded as an index of subnormal myocardial dependability. The majority of patients with fits do not die from fits.

I think we may sum up by saying that when we visualize a patient with block we must visualize a person who possesses a narrow margin of myocardial reserve. Standstill may kill him. Myocardial failure is more likely to.

It will probably be asked if the use of the electrocardiograph is entirely necessary in treating patients with heart block. The answer of course is "No." Many are being treated comfortably and efficiently without the aid of electrocardiography. This being at once granted it is nevertheless true that many cases of early delayed conduction have been missed, which later showed progressive increase. I have seen physicians and one or two otherwise good students who in the presence of a bradycardia had not even considered block. Careful observations and treatment might have averted some catastrophes. One may misinterpret signs, one may on bad days fail to appreciate the presence of these signs, one may on rush days fail to even look for them. In questions of doubtful conduction the electrocardiograph is our most accurate and dependable diagnostic aid and I think that in the not too distant future the portable EK machine will be probably considered part of the equipment of the physician who practices internal medicine and certainly of the one whose preference is the treatment of cardiovascular disease.

HYDRONEPHROSIS DUE TO AN ANOMALOUS BLOOD VESSEL

Report of a Case

S. G. LENZNER, M.D.*
PROVIDENCE, R. I.

Obstructive hydro-ureteral angularity, according to Hinman[1], can either be an acquired or congenital condition depending upon whether the primary obstruction is in the lower or upper portions of the ureter. In this report we are not concerned with the common types of hydro-ureter or hydronephrosis due to obstruction of the lower ureter. Hydronephrosis, however, due to a congenital cause, such as obstruction in the upper ureter, or at the uretero-pelvic junction, the common cause of which is an aberrant blood vessel, merits reporting.

Aberrant renal vessels are said to occur much more frequently than generally accepted. A complete historical and bibliographical review will not be attempted here as MATHE'[2] has fully and admirably covered the subject. On the Continent, DUVAL and GRE'GOIRE[3] reported their exhaustive study of the role of aberrant vessels in the pathogenesis of hydronephrosis in conjunction with LEGEAU et al[3], in which they give credit to BOOGARD, who reported the first case of this nature in 1857. In this country, EISENDRATH[4,5] not only summarized and described the different types of accessory arteries but brought up to date in 1920 all the cases reported in the literature from all authors.

Early recognition of these cases is of considerable importance if renal tissue is to be conserved. Only conservative surgery can be done in the early cases as shown by FENGER[6], who recognized this condition early, and carried out his classical conservative operation for valve like formations of the renal pelvis. JUDD[7] has modified this original operation by an ingenious procedure. However, advanced hydronephrosis always results in complete destruction of the kidney necessitating nephrectomy. Therefore, hydronephrosis should be considered in all cases presenting genito-urinary symptoms.

From an etiological standpoint, the majority of observers is of the opinion that anomalous vessels are a doubtful factor in producing obstruction at the uretero-pelvic junction. Thus, to quote from PANNETT[8], "The urenephros forms first and the descending pelvis extending downward carries the commencement of the ureter with it, thus bringing about the kinking over the abnormal vessels." Fig. 1 (after LAWSON), shows such an early case. KELLY[9] also was of the same opinion. MATHE'[2] has given in his complete paper the mechanism of gradual obstruction, due to various mechanical factors arising from the presence of the aberrant vessel. The operative findings of the case herein reported bears out the above facts to a striking degree.

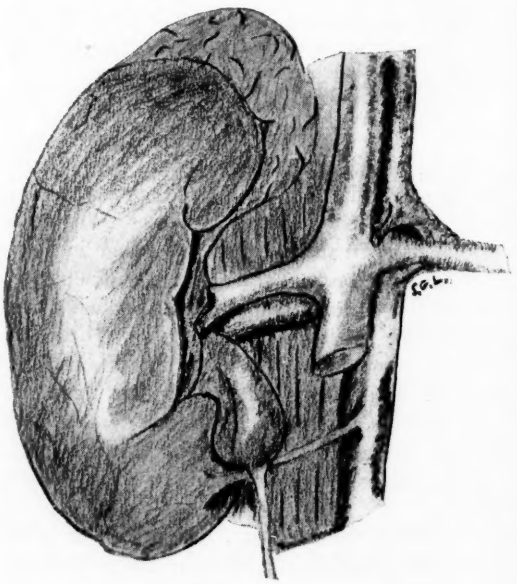

FIG. 1. AN EARLY CASE
Relation of vessel to ureter with dilatation of pelvis of kidney. (After Lamson.) Redrawn by the author from Surgical Clinics of N. America, Dec. 1928.

In this type of case, the question of treatment resolves itself into one of two methods. Many operators have tried conservative plastic operations on the pelvis with object in view of preserving a fair amount of parenchyma, but where hydronephrotic atrophy was progressive, a secondary nephrectomy was the rule. This clinical observation is confirmed by the experimental work of HINMAN[10], who found that the obstructed kidney would continue towards progressive atrophy in the presence of a compensatory hypertrophy of the

*From the Department of Surgery, Miriam Hospital. Read and discussed at Staff Conference, Miriam Hospital.

opposite kidney (unobstructed) even after the cause of the obstruction had been removed. In this particular case, so little normal parenchyma was found at the time of operation that nephrectomy was thought to be the wisest procedure.

Case Report

Miss R. K., hospital No. 2,583, school girl, aged 18. Entered the Miriam Hospital December 3, 1928, complaining of nausea and vomiting with dull pain in the left lumbar region of two years' duration. During infancy and early childhood, she had Measles, Whooping Cough, and Chicken Pox. At eight years of age, she was ill for six weeks with "Influenza." At first, patient had recurrent symptoms of nausea and vomiting following dietary indiscretions, such as too much pastry and sweets. Gradually she noticed the onset of pain with these "stomach attacks." Upon resuming a plain diet, the pain and nausea would disappear only to recur at no definite time. At no time would the pain extend down to the groin, but would be localized at the left free costal margin. At one time, her family doctor found albumin in her urine.

There is a negative history of frequency, dysuria, hematuria and nocturia. The urinary symptoms have been singularly absent in contrast to the clinical findings. There have been recurrent rises of temperature and chills concomitant with the gastric symptoms.

Abdominal examination reveals a definite bulging of the upper abdominal wall to the left of the median line. This bulging is fixed and does not move with respiration. Fluctuation is not present. A definite mass can be felt extending from the free costal margin to the umbilicus. Pressure in this area, however slight, elicits pain. Pulmonary type of breathing is present. The pain is most intense in the left costo-vertebral area. The right costo-vertebral area is not painful at all to the deepest pressure. The spleen cannot be felt. The area below the umbilicus shows no pathology. The groin shows the presence of small discreet lymph glands.

On December 6, cystoscopic examination was done. This revealed a normal right orifice with normal mucosa throughout. The urine collected from the right pelvis flowed in an intermittent stream, contained no pus nor bacteria, and eliminated 34 per cent. phthalein in thirty minutes. The appearance time of the dye was six minutes. The left orifice appeared punched out, similar to the opening of a diverticulum, but around the periphery of this opening, the mucosa was injected and showed inflammatory process of long standing. The catheter easily entered the orifice, but could not be advanced farther than 3 cm. because of some obstruction. No dye appeared from this side at the end of thirty minutes and no urine from this side could be obtained. Injection of 12.5 per cent. sodium iodide into the left orifice did not throw any shadow above bladder, showing catheter did not advance up the ureter. Subsequent injection of the right ureter showed a normal ureter and pelvis.

A few days later the patient felt the left sided pain had partially disappeared. Examination at this time showed a marked disappearance of the "tumor" with an increase in the urinary output. The phthalein output at this time for two hours

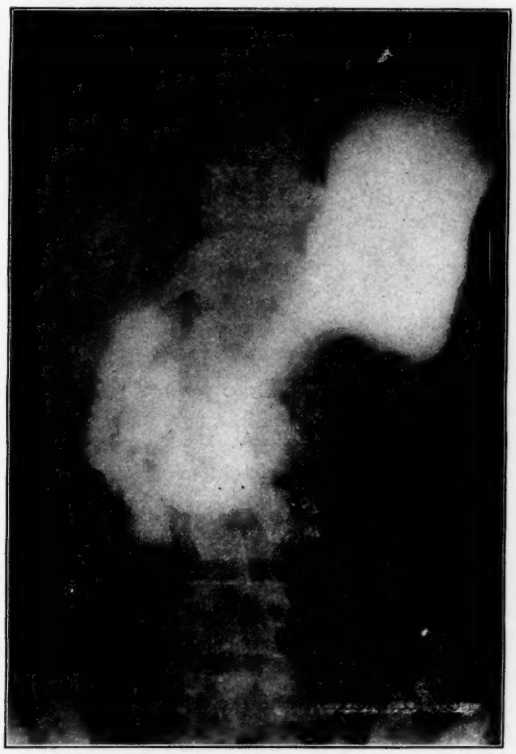

FIG. 2

Pressure of left-sided mass against greater curvature of stomach. Note smooth outline of retroperitoneal mass in its periphery.

was 75 per cent. Evidently irritation of the left ureter had set up a peristaltic wave, causing a temporary drainage from the left kidney pelvis.

A gastro-intestinal study was then made. Figure 2 shows how this mass compressed the greater curvature of the stomach, producing an hour glass contracture, and explaining the predominant gastric symptoms. The left dome of the diaphragm was not elevated and moved freely with respiration. No other pathology was found in the remainder of the gastro-intestinal tract.

Operation was performed on December 15, 1928 (Drs. McEvoy, Lenzner, Webber). A left oblique incision was made parallel with the quadratus

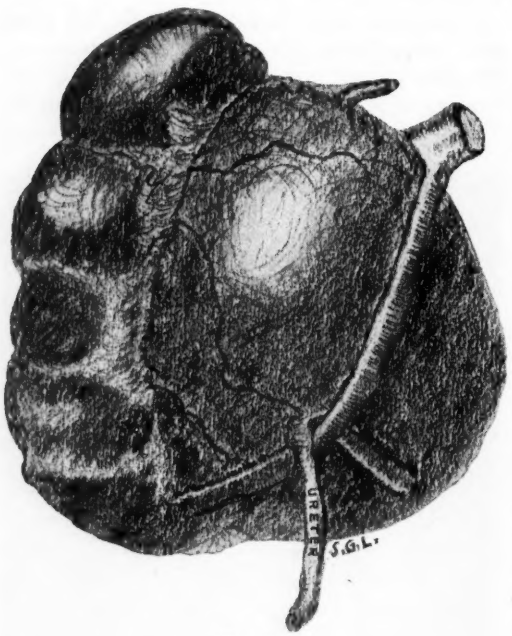

FIG. 3

Hydronephrotic kidney removed at operation—Kinking of ureter by anomalous vessel. Posterior view.

lumborum, extending high up into the costo-vertebral angle, avoiding ilio-inguinal and ilio-hypogastric nerves. Good exposure of the field was obtained. As the false capsule of the kidney was opened, considerable adhesions were found between capsule and kidney; the lower pole of the kidney was easily brought out of the wound. An anomalous blood vessel was then found crossing the uretero-pelvic junction posterior to the ureter coming from the abdominal aorta. There was a marked change in the size of the pelvis and the kidney. The pelvis was greatly ballooned out, very thick and fibrous in nature, above the anomalous artery (see Fig. 3). The ureter was of normal size below the artery. The kidney was greatly enlarged with great diminution of the parenchyma. At the upper pole, a large abscess could plainly be seen involving the entire cortex and parenchyma. The anomalous artery was then doubly tied and cut. The pedicle of the kidney was isolated, double clamps applied, two sutures No. 2 Chromic gut applied, the pedicle cut, and kidney removed. One cigarette drain was placed in the bed of the kidney. The wound was then closed in layers, suturing lumbar fascia and muscles with No. 1 chromic catgut.

Gross Pathology: The kidney weighed 189.6 gms. The greater part of the parenchyma was destroyed; the center of the kidney was entirely replaced by the very large pelvis, which was a very thick and fibrous structure. The upper pole was the seat of a large abscess. The length of the kidney was 13.75 cm.; width of the pelvis 7.5 cm. and the width of the kidney parenchyma itself was 4.4 cm. On cut section, the capsule stripped with difficulty, the cortex was greatly thickened and replaced in entirety by connective tissue. The pyramidal area was thinned out, a very few normal appearing pyramids of faintly pink color were seen; the columns of Bertini largely replaced by connective tissue deposition. The lesser calyces were clubbed and lengthened. Also the larger calyces were greatly dilated. Attached to the lower pole of the kidney was the ending of an anomalous artery running directly across the pelvis to the uretero-pelvic junction. The diameter of the ureter was normal below the artery.

Microscopical examination revealed the following (Dr. Kennison):

Low Power: The capsule is somewhat thickened and shows marked round cell infiltration and deposition of fibrolastic tissue. The glomeruli are shrunken. Bowman's capsules are distended with some exudate present in many of them. The ascending and descending Henle loops are collapsed so that the epithelial walls are in contact with each other. The connecting tubules, on the other hand, are widely distended without exudate to any extent. In several areas there is definite cystic formation displacing the parenchyma.

High Power: Various sections show some moderate differences in appearances. In general there has been a very marked degeneration and destruction of the normal cellular elements of the entire pyramidal region of the kidney. In some places this has gone on to total destruction and entire wiping out of cellular landmarks. Most of the glomeruli are shrunken and are undergoing chronic inflammatory changes. In some instances there have been shedding of normal epithelial cells into the periglomerular region. The blood vessels show flattening of intima with obliteration of all coats by fibroblastic tissue and in the larger radicles, passive congestion is very marked. Diagnosis: Degeneration, cloudy swelling as the result of obstruction, Hydronephrosis.

The postoperative convalescence was smooth and uneventful. The patient was discharged from the hospital on January 12, 1929. Follow up at present writing shows no genito-urinary symptoms and the renal dye function is 60-70 per cent. for two hours for the right kidney.

Summary

Hydronephrosis secondary to aberrant vessels is characterized by intermittent, regular attacks of renal pain, occurring over a period of months or years. Genito-urinary symptoms may be entirely lacking in these cases.

In the early type of case, conservative measures are the rule, but where a large portion of the kidney parenchyma has been destroyed by the pressure of the increasing size of the pelvis, secondary operations of nephrectomy must always be done because atrophy is progressive in nature. In this particular case, relief of the obstruction by division of the obstructing artery, would have given no assurance of complete relief of symptoms even if a plastic operation was done on the pelvis of the kidney.

Therefore early recognition of this clinical entity on the part of the clinician and surgeon will save many kidneys.

Acknowledgment is here made with thanks for the constructive criticism of Dr. McEvoy in the study of this case and the care on the part of Drs. Gerber and Alberts in their roentgenological examination.

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cance of compensatory hypertrophy and disuse atrophy to

THE FUTURE OF SURGERY

In the survey of the future of surgery, Lewis Hugh McKinnie, Colorado Springs, Colo. (*Journal A. M. A.*, April 6, 1929), asserts that men imperfectly qualified, even assuming a liberal standard, are doing surgery in every community. Students, under present conditions, cannot be adequately trained for the diverse activities that they will inevitably assume after graduation. The public is not safeguarded by the present diploma from a medical school, however well accredited it may be, since it seems to imply qualifications as a surgeon or specialist, which it cannot guarantee. The financial burdens devolving on the surgeon-to-be are so heavy that they seriously impede adequate training. He says the growing strength of the university and its ascendency in medical education suggests: (1) the graduate school of medicine as the coming standard; (2) the separation of the special fields of medicine prior to the granting of degrees, and (3) university supervision of postgradute study leading to a special degree.

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The R. I. Medico-Legal Society—Last Thursday—January, April, June and October. Roy McLoughlin, President; Dr. Jacob S. Kelley, Secretary-Treasurer.

EDITORIALS

THE AMERICAN COLLEGE OF PHYSICIANS

The program at the recent meeting of the American College of Physicians in Boston was probably the best that society has ever held. The society seems to have taken a distinctly new lease of life and most fortunately chose a city where clinical and teaching facilities were unexcelled.

The clinics at the various hospitals were most interesting and subjects presented were very timely and well chosen. Best of all the men selected to read the papers and to conduct the clinics were doctors who can make meetings interesting.

The selection of the city of Boston for this year's gathering was advantageous to Rhode Island doctors because many of them will not be able to attend the convention of the American Medical Association in Portland, Oregon, later in the year. It might be well for the American College of Physicians to choose for its meeting a city in another part of the country from that selected by the American Medical Association.

WHEN THE DOCTOR IS THE PATIENT

Many times in the course of his practice the average physician is called upon to care for a sick colleague. While he usually feels a real satisfaction in the knowledge of the confidence thus placed in him by his fellow practitioner it is the truth that he often undertakes the special responsibilities which his task involves with misgivings. This is not because he fears lest his patient with his intimate acquaintance with disease, will be critical of his methods or doubtful of his judgment but because, on the basis of previous experience in the case of members of his own profession, he knows that the doctor often makes a very unsatisfactory patient. This certainly should not be the case. No one should know as well as does the practicing physician how a patient should deport himself during an illness. The doctor, then, who spends his life ministering to the physical ills of others, trying perhaps, with a bit of impatience at times, to show them how to bear their troubles with equanimity, and who then, when his own turn has come, cannot show that fortitude and self control that he has expected of his patients, is a sorry spectacle. As has been so aptly said "the doctor who prescribes for himself has a fool for a patient" and yet one part of his treatment he alone can control, that is the mental attitude which he maintains towards his illness. It is true that his knowledge of disease raises in his mind specters as to complications and the ultimate prognosis which are mercifully spared the layman; while judgment and self control are often undermined by toxemia and weakness. Nevertheless native courage, a well balanced emotional control and a sound philosophy of life will carry him through many difficult situations if he has developed these qualities. One of the commonest mistakes that the sick doctor makes is that of trying to "run his own case." Until he has agreed that he is "licked" and is willing to pass entire control of the situation over to his physician and nurses the situation is almost hopeless. It is the experience of most of those who have physicians under their care that doctors make either the worst or the best of patients. Those who are masters of themselves and who can "practice what they preach" adding to their fortitude the factor of intelligent understanding co-operation, are patients to whom it is a pleasure and a privilege to bring professional aid. Such men are well equipped to care for the sick of their communities. It is difficult, on the other hand, to see how a man who lacks the ability to "stand the gaff" himself can qualify as a guide and helper to lead his patients safely in paths too thorny for himself to tread.

Personal experience is our greatest teacher fixing her lessons in our minds with a sureness that nothing can eradicate. What, then, can be more valuable in the education of a doctor than an illness successfully endured? Thus he may learn what his patients are really facing when he lightly orders for them a high enema, a hot pack or a dose of epsom salts, or when he decides to tap a chest or wash out an antrum. Thus, also, he can come to appreciate the value of nursing technique and the priceless ingredient of kindliness in bedside care. Above all he can prove to himself and to others what manner of man he really is and whether or not he is really qualified to play the part of physician by his own reaction to the acid test of illness.

THE MASSACHUSETTS GRADUATE COURSE IN CANCER

A very interesting experiment has just been concluded in our neighboring state of Massachusetts. The Massachusetts Medical Society has conducted a three day course in cancer for graduates. This was held with the assistance of the Massachusetts Department of Public Health and the local committee of the American Society for the Control of Cancer.

In these three days there was a very intensive and systematic survey of the entire subject of cancer from the pathological, surgical, medical, and radiologic aspects. Clinics, demonstrations and lectures were conducted at various hospitals, and arrangements were so handled that none of the demonstrations was overcrowded. At the same time everyone attending the course was able to obtain a comprehensive outline of the entire problem of malignancy.

This was the first time in the history of the state that any such project had been carried out. A similar intensive short course had been conducted a short while before in the state of Pennsylvania under the leadership of Dr. Jonathan Wainwright.

In the preliminary conferences before the course was arranged, those in charge felt that probably a fair representation of physicians would turn out, but they had no idea that the course would prove as popular as it did. The total registration for the course was 410, of which 211 were physicians and 199 dentists. The unusual and very large attendance of dentists was due to the fact that the first day of the course was devoted very largely to problems connected with malignancy of the mouth. The Massachusetts Dental Society cooperated very thoroughly in the arrangements. This willingness on the part of the dentists to learn something about cancer in and around the mouth is a very gratifying sign. It is upon the dentists that considerable reliance will have to be placed in the future for the early detection of oral cancer.

Altogether this novel experiment proved to be very gratifying to those concerned in arranging for the course, and of tremendous value to those who attended it. It is hoped that similar intensive courses will be available in the future for other states where there are proper facilities for clinics and demonstrations.

IN MEMORIAM

Resolution on the Death of Ransom H. Sartwell

WHEREAS: Almighty God has taken from us, Dr. Ransom H. Sartwell, Superintendent of the State Hospital for Mental Diseases, where his splendid qualities as a man, and his conscientious and efficient services as executive were known to all of us, and

WHEREAS: Dr. Ransom H. Sartwell was for many years a member of the Rhode Island Medico-Legal Society giving unselfishly of his time, his experience, his knowledge, and deep interest, in the welfare of our Society

THEREFORE: Be it resolved that the members of the Rhode Island Medico-Legal Society deeply regretting the loss of Dr. Sartwell to the State he served so well, and to this Society, and wishing to express our deep sorrow at his untimely death, order a copy of this resolution to be spread upon the records of the Rhode Island Medico-Legal Society, and a further copy to be sent to the family of our departed friend and brother.

Roy L. McLaughlin,
Wm. J. Harper,
Jacob S. Kelley, M.D.

NOTES

"COUNCIL PASSED"

Notification is being sent to the medical profession that Haley's M-O, Magnesia Oil has been accepted for N. N. R. of the American Medical Association. Henceforth the product will be known as Magnesia-Mineral Oil (25) Haley.

MISCELLANEOUS

THE EFFECT OF QUINIDINE SULPHATE ON VENTRICULAR TACHYCARDIA

Samuel A. Levine and Marshall N. Fulton, Boston (*Journal A. M. A.*, April 6, 1929), report their experiences with this drug in ten cases. The exact dosage is a variable matter and must be determined by trial and error. They usually begin with 0.3 Gm. of quinidine sulphate and repeat the dose every four to six hours, raising it 0.1 or 0.2 Gm. each dose or in urgent cases even more rapidly. In one instance it was necessary to give 1.5 Gm. five times within twenty-four hours before a normal rhythm was established, less than this amount being ineffective. After the return of the normal rhythm the patient is kept for a variable length of time on 0.2 or 0.3 Gm. three times a day or oftener if necessary. Quinidine sulphate has in most instances a specific effect in restoring a normal rhythm. Ventricular tachycardia occurs occasionally in patients who have no organic heart disease but more commonly in those with coronary thrombosis. Although quinidine does not have any effect on the other complications of coronary thrombosis, such as rupture of the heart or production of emboli, its beneficial influence on ventricular tachycardia is most dramatic and may be life saving. It is highly probable that ventricular tachycardia is of the nature of a circus movement like that of auricular flutter and auricular fibrillation, and that the effect of quinidine is similar in all these conditions.